A STUDY OF UPPER LIMB ISCHEMIA

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CERTIFICATE

This is to certify that this dissertation entitled ***"A STUDY OF UPPER LIMB ISCHEMIA"*** *submitted by* ***Dr. B. SARAVANAN*** *to The Tamil Nadu Dr.M.G.R. Medical University, Chennai is in partial fulfillment of the requirement for the award of M.Ch. degree Branch VIII (Vascular Surgery) and is a bonafide research work carried out by him under direct supervision and guidance.*

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DECLARATION

I, **Dr.B.Saravanan,** solemnly declare that dissertation titled **"A STUDY OF UPPER LIMB ISCHEMIA"** is a bonafide work done by me at Government Stanley Medical College and Hospital during January 2011 to December 2012 under the guidance and supervision of **Prof.Dr.S.R.Subrammaniyan, MS,Mch,FRCS** Professor and Head, Department of Vascular surgery, Government Stanley Medical College and Hospital, Chennai.

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CONTENTS

Introduction

Arterial diseases of the upper limb are relatively rare in comparison with those of the lower limb[1]. A large number of diseases can affect the arterial tree of upper limb. Upper extremity vascular reconstruction is much less common than the lower limb and it accounts for less than 5% of patients with upper limb ischemia[2]. The good collateral around the elbow and shoulder explains the reason why most of the chronic occlusive diseases are asymptomatic. Upper extremity vascular diseases can be classified based on anatomic location or on the basis of etiology.

The age distribution of patients is also broad with atherosclerotic affecting the older people and autoimmune affects the younger age group[14]. Occlusive disease can affect both the larger and smaller arteries. Subclavian is the most common larger artery involved in upper limb due to atherosclerosis whereas smaller vessels are involved most frequently due to arteritis. Aneurysms of the upper limb arterial tree are rare and may present with ischemia due to emboli and thrombosis[2].

Previous publications suggest that conservative management of patients with acute arm ischemia results in functional limb impairment in up to three-quarters of patients managed in this way and is therefore not advocated.

Our study deals with the various modalities and surgical techniques and its outcome that are commonly employed for revascularization of the upper limb include, thromboembolectomy, primary arterial repair with or without patch angioplasty, and autologous or venous bypass grafting[2]. These may occasionally be combined with concomitant cervical sympathectomy or cervical rib excision.

Aim of the study

The purpose of this study was to review the factors that will influence the outcome of upperlimb bypass and to identify those factors that are modifiable, and will help in improving the outcome of the procedures, improve the patency of the graft and limb salvage rate.

Review of Literature

The leading cause for upperlimb ischemia are autoimmune or connective tissue diseases such as rheumatoid arthritis, scleroderma, systemic lupus and others.

Causes of Ischemia:

The causes[1] of ischemia are as follows:

Large vessel	**Small vessel**
Atherosclerosis	Connective tissue diseases
Thoracic outlet syndrome	Myeloproliferative disorders
Arteritis (Takayasu's, Giant cell)	Buerger's disease
Fibromuscular disease	Hypersensitivity angitis
	Cold injury
	Henoch-Schonlein purpura
	Cytotoxic drugs
	Hypercoagulable states
	Arterial drug injection

Ischemia can also occur as a result of embolism from larger vessel to distal small vessel due to ulcerated plaque rupture or from aneurysms .

Diagnostic Evaluation

Evaluation of patients with ischemia begins with detailed history taking, physical examination and investigations

Detailed History :

The history should focus on the signs and symptoms of specific diseases[6]. For example in ischemia due to connective tissue diseases history of dry eyes, dry mouth and arthritis should be emphasized.

Most important is the history of trauma and history of exposure to virtating tools or of toxins.

Physical Examination :

The hands are examined for temperature, capillary refill, and ulcers and gangrene are noted. Fingers are examined for clubbing, sclerodactyly, telengiectasia, and nail beds for splinter hemorrhage . Emphasis is given for the muscle mass, strength, and sensation. Complete examination of pulses.

Blood Examination :

Erythrocyte sedimentation rate, C-Reactive protein, antiphospholipid antibodies, antinuclear antibody titer, and rheumatoid factor are done to screen for autoimmune diseases.

Investigation :

1. X-ray cervical spine both anteroposterior and lateral views to rule out cervical rib
2. Segmental pressure measurement in brachial, radial and ulnar arteries
3. Doppler waveforms
4. Digital plethysmography
5. Cold sensitivity test
6. Duplex examination
7. Arteriography

Segmental Pressure :

Upper arm and forearm blood pressures are measured with the help of sphygmomanometer and hand Doppler. In patients with work claudication pressures are obtained at rest and after 5 minutes of exercise.

Doppler waveforms :

Doppler velocity flow to look for continuous wave is used for assessing arm perfusion when pulses are not palpable. The larger arteries from subclavian to the smaller arteries like digital arteries can be checked for the presence and quality of Doppler signal like triphasic, biphasic, monophasic or dampened signal.

Digital plethysmography :

Digital plethysmography[2] flow patterns are useful in the evaluation of digital ischemia and vasospastic disorders. Diminished or flat pulse volume recordings confirm the reduction in digital flow. Pulse volume recordings are a useful tool in assessing the digital flow after the initiation of treatment and in follow up of the patient.

Duplex ultrasonography :

It helps in excellent visualization of the upper extremity vascular tree. It is an useful tool both as B-mode and to assess the velocity thro the segment of the vessel of study. The main drawback[7] is to study the proximal subclavian artery as it is covered by the clavicle.

Arteriography :

Aaortography[5] is the gold-standard examination to delineate the extent and location of great vessel pathology. It also helps to study the four-vessel runoff so that inflow can be taken from the carotid vessel.

The latest digital subtraction techniques can be done with decreased contrast and with less time. The drawbacks is of intraarterial contrast, the risk of stroke due to catheter manipulations, the nephrotoxicity of the contrast agents, and the possibility of missing irregular lesions due to inability to visualize them through the column of contrast.

Magnetic resonance angiography (MRA) :

This is noninvasive and is done without contrast or less toxic gadolinium is used for the study. The sensitivity[1] and the specificity at detecting a hemodynamically significant stenosis was 90% and 96%, respectively. The technology and techniques for both obtaining and analyzing data are improved with high definition hence MRA is likely to replace conventional arteriography of the aortic arch and its branches.

MRA is more accurate in diagnosing and dileatening plaque morphology and ulceration, MRA is more sensitive than conventional angiography, in diagnosing lesions which are obscured by the column of

contrast as in conventional arteriography. The draw back of gadolinium as a magnetic resonance contrast agent is the potential to produce nephrogenic systemic fibrosis in patients with a glomerular filtration rate of less than 30 ml/min.

Computed tomography angiography :

This is also a noninvasive technique like MRA . This technique can also be used to image the aortic arch and branch vessels.

In summary, the radiographic evaluation of a patient suspected of having symptomatic upper limb ischemia should begin with duplex ultrasonogaphy. Further evaluation of the lesion that is clearly responsible for the symptom or is suggestive of a proximal stenosis, should be done to image the aortic arch its branches and the great vessels. If clinical suspicion for significant proximal disease is low, an MRA or a computed tomography angiogram beginning at the level of the aorta should be obtained. A normal study excludes significant pathology. If suspicion of a lesion that will require intervention is high, a conventional intraarterial angiogram is recommended, especially if a percutaneous intervention at the time of angiography is being considered.

To evaluate a patient with upperlimb ischemia, computed tomography angiography or MRA before intervention could be helpful in guiding an endovascular intervention.

Atherosclerotic occlusive Disease

Atherosclerosis[3] may cause upper extremity ischemia through a variety of mechanisms. The most common is occlusive disease, with involvement of the origin of the subclavian artery being a common finding in patients with significant atherosclerosis. Aneurysms[8] of the upper extremity arteries are rare but can occur in the subclavian or axillary arteries. Patients with diabetes or renal failure, or both, particularly those undergoing dialysis, may have accelerated atherosclerosis and not infrequently have involvement of both large and small arteries of the upper extremity that can lead to significant hand ischemia.

The pathophysiology[2] of atherosclerotic lesions is different given the potentially irregular nature of the luminal surface. For those lesions requiring surgical intervention, it is twice as likely that an embolic event is the cause of the presenting symptom.

Upper limb ischemia due to atherosclerosis commonly presents with hand and finger ischemia and more so in patients with end-stage renal disease. The etiologic backgrounds of this disease process are multiple and include thrombosis, accelerated atherosclerosis, and diffuse arterial calcification. The origin of this condition may well involve atherosclerosis,

but there is a well-described association with calciphylaxis. Manifestations range from digital pain at rest and ulceration to gangrene.

The evaluation is by noninvasive studies such as pulse volume recordings or duplex ultrasound, followed by magnetic resonance or computed tomographic angiography. Occlusive lesions will be located in the axillary, brachial, or forearm arteries. Each requires separate treatment strategies.

Management :

Endovascular Treatment

Endovascular therapies[5] for the treatment of arterial occlusive disease have increased in over the last decade with the improvement in catheter, balloon, and stent technology. The majority of lower extremity revascularizations in large vascular surgery practices are now done percutaneously. However, the upper extremity has not shared the same paradigm shift, possibly because of the infrequency of interventions or the causes of the arterial disease. Most institutional reviews that describe treatment of occlusions in the axillary, brachial, radial, and ulnar arteries still involve surgical bypass or embolectomy.

Surgical Treatment

Bypass and tunneling :

Autogenous vein is the conduit of choice for upper extremity reconstructions. The great saphenous vein is preferable, although use of in situ cephalic vein has been described. After systemic heparinization, the venous conduit is excised and its side branches ligated with fine polypropylene suture or silk ties. The vein is distended with a solution of dextran, heparin, and papaverine (500-mL bag of dextran 40 + 120 mg papaverine + 1000 units heparin). The excised conduit may be used in either reversed or orthograde (nonreversed) orientation, depending on the taper of the conduit. If orthograde orientation is used, the proximal anastomosis is performed, the conduit is distended, and the valves are rendered incompetent by passage of a retrograde Mills valvulotome. Prosthetic reconstructions are also used but have lower patency rates than do venous bypasses .

Bypasses originating from the axillary artery are preferably tunneled anatomically along the axis of the axillary and brachial arteries because they are then less prone to movement or distortion. They may also be positioned anteriorly in the subcutaneous plane. However, superficial bypasses are more prone to distraction injuries from forcible abduction of the shoulder. Therefore, redundancy is paramount to avoid harming the conduit.

Bypasses based on brachial artery[13] inflow are most often tunneled in the subcutaneous plane. This facilitates physical examination for appropriate evaluation of bypass patency, ensures surveillance of the bypass with duplex if readily available, and avoids manipulation of the rich forearm nerve network. Alternatively, if good-quality basilic or cephalic veins are present, an in situ bypass may be performed.

In the case of distal radial artery reconstructions[11], the graft is tunneled over the anatomic snuff-box onto the dorsum of the hand, between the thumb and index finger, to join the deep palmar arch . In the case of ulnar artery reconstruction, the course of the vein graft is more direct and it passes superficial to the flexor retinaculum at the wrist to join the superficial palmar arch.

Transbrachial Embolectomy

The majority of arm emboli are cardiac[2] in origin (75%). The most common site for emboli is the brachial artery (60%), followed by the axillary artery (26%). In situ thrombosis accounts for only 5% of episodes of arm ischemia. Usually, there is an underlying embolic source such as atrial fibrillation. The majority of brachial embolectomies are performed under local anesthesia with monitored anesthesia care.

When an embolectomy is planned, several considerations are essential. If preoperative imaging (usually duplex ultrasound) has demonstrated occlusion of the distal brachial artery, it is important to expose the origins of both forearm arteries because the embolectomy catheter must be passed down each artery. If the catheter is passed blindly down the brachial artery, it will most likely travel down the radial artery. This will probably re-establish flow to the hand in the majority of patients. However, it may fail to restore adequate flow if the ulnar is the dominant arterial blood supply or if the catheter passes down the common interosseous artery, which does not provide any direct flow to the hand. We usually make a vertical arteriotomy in the brachial artery. Clot, frequently encountered at the bifurcation, can readily be removed. The brachial artery may be pulseless, indicative of an embolus lodged more proximally. However, one concern with proximal passage of an embolectomy catheter is dislodgment of debris that may embolize the vertebral artery near the origin of the subclavian artery. If there is any concern, contrast-enhanced angiography is warranted to define the anatomy. Once inflow is established, a size 2 or 3 embolectomy catheter is passed distally down each forearm vessel. The arteriotomy can be closed primarily with running fine polypropylene suture if the artery is sufficiently large. If there is any doubt, the artery should be closed with a

vein patch. Usually, a segment of vein can be harvested from the antecubital fossa.

After closure, an intraoperative continuous wave Doppler probe should be applied to each artery to ensure the presence of adequate flow. Completion angiography is at the discretion of the surgeon but should be performed if the hand still appears ischemic, especially if extensive thrombus has been extracted from forearm arteries. Occasionally, embolization may have been a chronic process and resulted in distal arterial occlusion that cannot be completely resolved with embolectomy, but arteriography can be used to detect residual fresh thromboembolic material. If there is any suspicion of an inflow lesion, intraoperative arteriography can be performed by either the femoral or brachial route to diagnose and treat such lesions.

Postoperative Management and Follow-up

Postoperatively, surveillance with pulse volume recordings or Doppler segmental pressure measurements can be used to document the adequacy of the bypass. Duplex ultrasonography[10] is used to determine the patency of the reconstruction and allow early detection of stenoses during surveillance of the venous conduit. Anticoagulation is essential after

embolectomy. Recurrent embolization occurs in a third of patients after successful embolectomy if systemic anticoagulation is not instituted.

Complications

Major complications[16] that may occur after upper extremity arterial surgery include brachial plexus and median or ulnar nerve injury. Such injury is usually a result of traction and should be avoided by careful dissection during operative exposure. The use of electrocautery should be minimized to lessen the chance of direct thermal injury to the underlying nerves at all levels.

As with any arterial bypass, graft thrombosis can also occur. Some series found female smokers and patients with longer bypasses crossing multiple joints to have the lowest patency. An inflow lesion in the subclavian artery can also cause acute arm ischemia (e.g., ulcerated plaque, thrombosed subclavian artery aneurysm, or arterial thoracic outlet syndrome). In such situations, even a technically perfect brachial embolectomy will fail to restore normal hand perfusion. Moreover, if the arteries are relatively free of atherosclerotic disease, embolectomy of the distal radial and ulnar vessels may result in arterial spasm. This problem is more commonly seen in women and children. However, it is a diagnosis of exclusion, and the first priority must be removal of retained embolic

debris. If spasm is identified, topical or intra-arterial vasodilators such as papaverine, warm saline irrigation, and time should relieve the constriction and result in an adequately perfused hand.

Takayasu's Arteritis

The etiology of takayasu's arteritis is multifactorial[2]. The arterial inflammation is an immune-mediated response with a genetic predisposition. The histopathological changes consist of plasma cells and lymphocytes within the adventitia and media. Giant cells can form within the media, and elastic fibers can be identified within these lesions. After degradation of the media elastic fibers, it becomes fibrotic. This fibrosis results in the long, tapered stenosis of the affected vessels. The intimal injury[1] and subsequent proliferation or thickening results from damage in the vaso vasorum and this intimal damage contributes to development of stenoses and occlusions.

The anatomical distribution of disease in Takayasu's arteritis varies depending on the ethnicity, location, or both of the study population. Japanese populations have a higher incidence of isolated supraaortic and aortic arch involvement. The frequency with which each vessel may be involved, is as follows.

Anatomical Distribution of Disease in Takayasu's Arteritis

ARTERY	Degree of Stenosis	
	≥50%	100%
	No. (%)	No. (%)
Innominate artery	10 (77)	5 (38)
Right common carotid artery	9 (69)	4 (31)
Left common carotid artery	8 (62)	4 (31)
Right subclavian artery	8 (62)	5 (38)
Left subclavian artery	12 (92)	10 (77)

Distribution of hemodynamically significant (>50%) stenoses and complete (100%) occlusions in 13 patients with symptomatic great vessel disease from Takayasu's arteritis.

Takayasu's arteritis[6] is most common in females. The Initial presentation is before the age of 40. The symptoms include fever, malaise,

myalgia, limb claudication or pain overlying an involved artery. Hypertension and its sequelae are the most common presentation. Hypertension can be from renal artery stenosis, aortic coarctation, or carotid involvement with alteration in the usual baroreflex. Neurological symptoms related to the hypertension can include headache, visual changes, stroke, or even encephalopathy. Lightheadedness, dizziness, photophobia, visual disturbances, and clinical subclavian steal syndrome can all occur. Progression of the diseases causes stenosis or occlusion of the subclavian, innominate, and carotid arteries.

Patient present with signs of absent pulse in one or both upper extremities. Early in the course of disease, there may only be discrepancies in blood pressures between the arms or between the arms and the lower extremities. Subclavian or carotid bruits can often be heard, as well as intraabdominal bruits. Carotid pulses may also be blunted or absent.

Diagnosis :

There are many diagnostic criteria[1] :

American College of Rheumatology Criteria for Takayasu's Arteritis[1]

Criteria	Definition
Age at disease onset	Symptoms/findings of Takayasu's arteritis before age 40 years
Extremity claudication	Lower or upper extremity muscle fatigue during exercise
Diminished brachial artery blood pulse	Unilateral or bilateral diminished pulse
Blood pressure (BP) difference	>10 mm Hg difference in measured systolic BP between upper extremities
Bruit	Bruit over one or both subclavian arteries or aorta
Angiographic abnormalities	Narrowing or occlusion of aorta, primary branches, or large arteries in proximal extremities; must not be secondary to atherosclerosis, fibromuscular dysplasia, or other causes; usually focal or segmental

In the presence of two major criteria, one major criterion and two minor criteria, or four minor criteria was found to have a sensitivity of 92.5% and a specificity of 95% in a group of Indian patients and 96% sensitivity and 96% specificity in a group of Japanese patients.

Modified Diagnostic Criteria for Takayasu's Arteritis[1]

Criteria	Definition
Three Major	
Left mid–subclavian artery lesion	Severe stenosis or occlusion present in midportion from 1 cm proximal to left vertebral artery origin to 3 cm distal to origin
Right mid–subclavian artery lesion	Severe stenosis or occlusion in midportion from right vertebral artery origin to 3 cm distal to origin
Characteristic signs and symptoms ≥1 month's	Includes limb claudication, pulselessness or pulse difference in limbs, unobtainable or significant blood pressure difference

Criteria	Definition
duration	(>10 mm Hg systolic) in limb, fever, neck pain, transient amaurosis, blurred vision, syncope, dyspnea, palpitations
Ten Minor	
Elevated erythrocyte sedimentation rate (ESR)	Unexplained persistent ESR >20 mm/hr at diagnosis or in patient history
Carotodynia	Unilateral or bilateral tenderness of common carotid artery on palpation; must differentiate from neck muscle tenderness
Hypertension	Persistent elevation of blood pressure >140/90 mm Hg (brachial) or 160/90 mm Hg (popliteal)
Aortic regurgitation/annuloaortic ectasia	By auscultation, echocardiography, or angiography

Criteria	Definition
Pulmonary artery disease	Lobar or segmental artery occlusion or equivalent; stenosis, aneurysm, luminal irregularity, or any combination in pulmonary trunk or pulmonary arteries
Left mid–common carotid artery lesion	Severe stenosis or occlusion in midportion, 5 cm long, from a point 2 cm distal to origin
Distal brachiocephalic trunk lesion	Severe stenosis or occlusion in distal third
Descending aortic lesion	Narrowing, dilatation or aneurysm, luminal irregularity, or any combination involving thoracic aorta; tortuosity alone is insufficient
Abdominal aortic lesion	Narrowing, dilatation or aneurysm, luminal irregularity, or any combination involving abdominal aorta; tortuosity alone is insufficient

Criteria	Definition
Coronary artery lesion	Narrowing, dilatation or aneurysm, luminal irregularity, or any combination before age 30 years absent atherosclerotic risk factors such as hyperlipidemia or diabetes mellitus

Investigation :

The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are the most common markers used. serum interleukin-6 (IL-6) and RANTES(regulated upon activation, normal T-cell expressed, and presumably secreted) levels are increased in patients with TA. Also MMP-2, MMP-3 and MMP-9 levels were elevated in patients with TA.

Radiologic Evaluation :

Noninvasive[7] methods such as ultrasound, computed tomographic angiography (CTA), and magnetic resonance angiography (MRA) have been used to assess the mural changes of the arterial wall seen in TA before the development of stenotic or aneurysmal disease.

Findings on CTA may include high density and calcifications of the aortic wall on precontrast images, a thickened wall with enhancements in the arterial and venous phases, and a low-attenuation ring in the venous phase . CTA is also useful to assess the extent of disease involvement owing to its ability to assess mural inflammation.

MRA is also useful for the noninvasive assessment of patients with TA. Spin-echo MRA depicts early arterial wall thickening similar to CTA. Although MRA accurately depicts approximately 98% of arterial involvement in TA, 2% of stenotic arteries are overestimated as occluded. Hence MRA is not feasible when determining treatment response.

Positron emission tomography (PET) is also used to evaluate the inflammatory process associated with takayasu's arteritis. . Quantification of ^{18}F-fluorodeoxyglucose (^{18}F-FDG) uptake can be used to evaluate active vascular inflammation before the development of hemodynamically significant stenoses. It is also useful to identify the disease activity and also in accurately monitoring treatment efficacy . F-FDG PET strongly correlates with the response to medical therapy and may prove useful for managing patients whose serum inflammatory markers remain elevated despite inactive disease.

Digital subtraction angiography[2] remains the “gold standard” for evaluation of the vascular lesions associated with TA. Panangiography depicts the extent of disease, which ultimately correlates with disease severity, and is useful in preoperative planning. Lesions characteristic of TA include stenoses, either short and segmental or long and diffuse; aneurysmal dilatation, either fusiform or saccular; or a combination of the two. Comparison of direct aortic blood pressure measurement at the time of angiography to measurements in the upper and lower extremities is important owing to the nature of stenotic lesions and their interference with extremity cuff pressures. Finally, the classification of TA is based on arteriographic findings and correlates with clinical manifestations and prognosis.

Treatment :

Transsternal reconstruction for aortic branch occlusion has been the gold standard[2]. Carotid-subclavian bypass is the treatment of choice for isolated disease. Remote, non-anatomical bypass like axilloaxillary, carotid-carotid bypass are the options for patients who are at risk for transsternal or cervical bypasses.

PTA and stenting[5] is gaining popularity as first line therapy for proximal lesions because of its low morbidity and mortality but the long term patency of these endovascular approaches remains unclear.

Upper Limb Thromboangitis Obliterans

Buerger's disease (thromboangiitis obliterans) describes a clinical syndrome characterized by the occurrence of segmental thrombotic occlusions of the small- and medium-sized arteries. Though the lower extremities are most often involved the upper extremity involvement with resulting Raynaud's syndrome and digital ischemia occurs in as many as 50% of these patients. Ischemic digital ulcerations are common.

The diagnostic criteria[2] include age less than 45 years, tobacco abuse, exclusion of other diseases with similar clinical findings, normal arteries proximal to the popliteal or brachial arteries, and documentation by objective means of digital arterial occlusion.

The pathological lesion of Buerger's disease, in the acute stage, is one of neutrophilic inflammation with preservation of the internal elastic lamina. Other distinguishing features include preservation of the arterial wall and lack of vascular wall calcification, aneurysms, and atheroma. Lesions eventually develop a predominance of mononuclear cells and occasional giant cells. Late lesions are characterized by intense

perivascular fibrosis. The cornerstone of treatment of Buerger's disease is cessation of smoking. With this, most lesions heal with simple conservative therapy.

Scleroderma

The most common connective tissue disease in patients with Raynaud's syndrome is scleroderma. Scleroderma (progressive systemic sclerosis) is a generalized disorder of connective tissue, the microvasculature, and the small arteries. The name scleroderma is derived from the Greek for "hard skin." Scleroderma has a female-to-male ratio of about 3:1. Cutaneous involvement in scleroderma is almost universal. The clinical course of scleroderma is characterized by progressive scarring and small vessel occlusions in the skin, gastrointestinal tract, kidneys, lungs, and heart. Raynaud's syndrome[2] is present in 80% to 97% of patients with scleroderma. The syndrome usually begins as the vasospastic type and progress to the obstructive type over time in a subgroup of patients.

Scleroderma patients may be divided into those who present with involvement of primarily the skin of the hands, forearms, and face (limited involvement group) and those who present with diffuse cutaneous and visceral involvement (diffuse involvement). Members of the first group, which represents about half of the total, often present with finger swelling

and longstanding Raynaud's syndrome. Perhaps because of the usual delay in presentation to a physician, these patients in the limited involvement group are older than those in the diffuse involvement group. The limited involvement group appears to have a relatively benign course with prolonged survival in comparison to the diffuse involvement group. This limited cutaneous involvement syndrome has been termed the CREST (for calcinosis, Raynaud's syndrome, esophageal dysmotility, sclerodactyly, and telangiectasias), although many patients with limited involvement do not manifest all these features.

Patients with scleroderma may also be divided into subgroups based on the presence or absence of various antibodies. Patients with limited involvement often have a positive anticentromere antibody, while those patients with diffuse involvement are more likely to have antibodies to topoisomerase I (anti-Scl-70).

While the pathophysiological[1] mechanisms of scleroderma are still being elucidated, the damage seen in scleroderma is likely mediated through cytotoxic antibodies to the endothelium. Histopathological features of scleroderma include a vasculitis of the small arteries with fibrinoid necrosis and concentric thickening of the intima with deposition of layers of mucopolysaccharides. The cellular infiltrates seen on histopathology are composed of T cells. Capillary abnormalities may be

seen in patients with scleroderma .These capillaries have thickened basement membranes and endothelial damage.

Systemic lupus erythematosus (SLE)

SLE have been reported in all age groups but is most common in young females, and only about 10% involvement in male patients. The pathophysiology of SLE remains unknown, and the damage to the affected organs appears caused primarily by immune complex deposition. Abnormalities in apoptosis have also been postulated in SLE.

The diagnosis[2] of SLE is made primarily clinically since the available laboratory tests in patients with SLE, such as the antinuclear antibody, while quite sensitive, are not specific. The clinical criteria include fevers, arthralgias, skin rash, Raynaud's syndrome, and nephritis.

The pathological lesions of SLE typically involve small arteries and capillaries and occur throughout the vascular system. The lesions in the small arteries are characterized by necrosis of all or part of the vessel wall, fibroblastic hyperplasia of the intima or media, and occlusive fibrin deposits. Raynaud's syndrome is one of the most common clinical manifestations of SLE, with as many as 80% of patients reporting this symptom. Some patients, develop significant digital arteriopathy with

resulting digital artery occlusion, digital ischemia, and in the extreme digital ulceration and gangrene.

Vibration Arterial injury

The finding of Raynaud's syndrome after long-term use of vibrating tools was first reported about a century ago. The patients in this group include stonecutters, welders or grinders in shipyards, timber fellers, and windshield replacement technicians in the auto-glass industry. This condition has been termed vibration white finger.

The pathophysiological mechanism[1] of vibration white finger is not known, but clearly kinetic energy imparted to the small vessels and nerves of the hand by vibrating tools with power in certain frequency bands is harmful. This appears to be a cumulative trauma disorder in that the damage accumulates over time without healing between exposures. Initially these patients present with vasospastic Raynaud's syndrome, but in the late stage, occlusion of digital artery occurs.

Thoracic outlet syndrome

The limited space and numerous important structures that must traverse the neck and chest areas on their way to the upper arm make the thoracic outlet an area like no other in the body. Although any number of

anatomical anomalies can predispose a patient to or directly cause compression to the neural, venous, or arterial structures within its confines, the normal anatomy itself leaves little room for stress positioning.

Thoracic outlet is the area from the edge of the first rib extending medially to the upper mediastinum and superiorly to the fifth cervical nerve. The clavicle and subclavius muscles can be pictured as forming a roof, while the superior surface of the first rib forms the floor. Machleder's[1] description of the thoracic outlet as a triangle with its apex pointed toward the manubrium is helpful in visualizing the three-dimensional orientation of the structures, as well as the dynamic changes that can lead to injury. In this model, the clavicle and its underlying subclavius muscle and tendon form the superior limb, while the base is the first thoracic rib. The point at which these two structures "overlap" medially can be pictured as the fulcrum of a pair of scissors that open and closes as the arm moves, potentially causing compression of the thoracic outlet contents.

Although most TOS symptoms are related to nerve compression, almost any structure that travels through the thoracic outlet can be involved . Moving medially to laterally, the clinician first encounters the exiting of the subclavian vein, usually positioned adjacent to the region where the first rib and clavicular head fuse to form a fibrocartilaginous joint with the

manubrium. Immediately lateral to the vein is the anterior scalene muscle, which inserts onto a prominence on the first rib. Lateral to this site is the subclavian artery so that the anterior scalene muscle lies between the subclavian artery and the vein, with the artery deep, lateral, and somewhat cephalad. The brachial plexus is the next structure encountered. The C4 to C6 roots are superiorly oriented, and the C7 to T1 roots inferior. As shall be seen, this arrangement has important implications for the symptom constellation in neurogenic TOS. Posterior and lateral to the plexus, generally a rather broad attachment of the middle scalene to the first rib is found. This is an area of particular importance during operative decompression of the thoracic outlet, for here the long thoracic nerve can be inadvertently injured as it travels to the serratus anterior muscle.

Pathophysiology

Once the normal anatomy of the thoracic outlet is appreciated, it becomes clear that any structure that intrudes into or limits the flexibility of this region predisposes the nerves, arterial structures, and venous structures to compression. In addition to the obvious compression to the contents of the thoracic outlet, histological and biochemical changes are observed in the involved tissues. These stress the uniqueness of the outlet's configuration and offer objective support to the chronicity of many of these injuries.

The cervical rib is the most obvious bony abnormality contributing to TOS, and autopsy studies indicate that roughly 0.5% of the population have this structure . Cervical ribs can be completely formed or rudimentary. In the latter case, a compressive band of tissue usually extends to the first rib. As they project from transverse processes, cervical ribs displace involved structures forward. The subclavian artery is particularly vulnerable to damage in this configuration.

Several other bony abnormalities are found in association with TOS. Posttraumatic changes following clavicular or first-rib fractures are commonly reported, with callous formation at the clavicle and pseudoarthroses of the first rib. Elongated C7 transverse processes are also occasionally seen. These changes can often be appreciated radiographically.

Most TOS cases are associated with some form of soft-tissue anomaly. the classification system of Roos, with 10 distinct anomalies and several subtypes observed intraoperatively are as follows.

Roos Classification System[1]

Type I	Incomplete cervical rib; band beneath T1 root attached to first rib

Type II	Abortive cervical rib with band to first rib
Type III	Accessory muscle between neck and tubercle of first rib; separates T1 nerve and the subclavian artery
Type IV	Large middle scalene compressing T1 nerve root; pins nerve to the vertebral body (ulnar symptoms)
Type V	Scalenus minimus muscle attaching to the first rib behind the scalene tubercle
Type VI	Scalenus minimus muscle attaching to the endothoracic fascia covering the cupola of lung
Type VII	Band extending from the middle scalene to the costal cartilage or sternum
Type VIII	Band from middle scalene under the subclavian vein; associated with Paget-Schroetter syndrome
Type IX	Web filling the inner curve of the first rib

Type X	Double band from cervical rib attaching to the cupola with a band to the costocartilage or sternum

Several other muscular anomalies exist, mostly involving thickened muscle branches encompassing nerves, for example, slips of anterior scalene between the C5 and the C7 nerve roots or even a bulky muscle anteriorly displacing the nerves. Reports have been made of these muscle fibers becoming incorporated into the epineurium of these nerves as well. One particularly interesting configuration is when a fibrous band stretches over the proximal C5 nerve. Muscular spasm of the neck and shoulder can then lead to direct compression, with upper cord symptoms.

In addition to these anatomical arrangements, hypertrophy of the normal musculature or tendons has been implicated in TOS. Another scenario is the weight lifter, often a young man, with hypertrophied scalene musculature. Anatomical studies have documented the compression of the subclavian vein by this muscle into the costoclavicular notch. This has clear implications for axillosubclavian vein thrombosis.

Trauma is generally implicated in the pathogenesis of neurogenic TOS, particularly localized to the neck and shoulder. Hyperextension, or

whiplash injuries, occurs in this patient population with some frequency. Neurogenic TOS can also be seen with repetitive motion–type injuries. This is termed the pectoralis minor syndrome, and the symptoms can be reproduced with an external compression of the pectoralis musculature.

As mentioned previously, histological and biochemical changes in the tissues are involved with TOS. Work by Sanders et al. demonstrated inflammation surrounding compressed nerve trunks. This inflammation could cause direct irritation of the nerve trunks or set up a perineural inflammatory process leading to vasospasm in the vasa nervorum. Histological changes in both the mesoneurium and the endoneurium of involved nerves have been described, and the local formation of edema and adhesions is often observed.

On the molecular level[1], several reports have noted the transition from type 2 to type 1 muscle fibers in TOS. Briefly, most skeletal muscle fibers are the quick-reacting type 2 fibers that have reactivity with phosphorylase and myosin ATPase. The slower, type 1 fibers have an increased oxidative capacity and a pronounced, slow, tonic, contractile pattern. Immunohistochemical analysis shows greater numbers of type 1 fibers in the anterior scalene muscle of TOS patients than in any other kind of tissue. The signaling required for this transition is not completely

understood, but it does provide a molecular backdrop to the gross changes seen in TOS.

Arterial

Subclavian artery compression can lead to several injuries, its presentation is the most varied of the three forms of TOS. Damage to the subclavian artery itself can lead to anywhere from a small stenosis to aneurysm formation or complete occlusion. Each of these can then have its own sequelae secondary to embolization or thrombosis or the extremely rare rupture of a subclavian aneurysm.

These patients presents with ischemic conditions of the hands, with paronychial ulcers or fingertip gangrene. If the subclavian artery is completely occluded, patients may present with early fatigue of the involved side. This can be in the form of crampy pain with exercise and has led to the term arm claudication.

Diagnosis

It is important to point out from the outset that no generally accepted battery of tests must be performed to confirm the presence of TOS. Diagnosis include a physical examination consistent with the symptoms,

cervical films to rule out disc disease, and a chest radiograph to visualize any bony abnormalities.

History and Physical Examination

A detailed history should be taken from the patient, including any injuries and the patient's occupation. In conjunction with a good history, most cases of TOS are diagnosed on the basis of physical examination. This includes the general appearance of the patient and other signs of symptom impact. Serratus anterior atrophy is occasionally present with TOS, as demonstrated by a winged scapula. Deep-tendon reflexes, grip strength, and pulses should be routinely assessed. Palmar hyperhidrosis should be noted if present.

Patients with TOS generally do not have obvious muscle atrophy; rather, they have nearly normal gross baseline sensory and motor exams. Initial palpation of the structures of the chest wall, cervical area, and shoulder can be useful before undertaking provocative testing. The region overlying the anterior scalene muscle is often exquisitely tender in the face of brachial plexus entrapment or irritation. In addition, percussion of the clavicle can reproduce pains and paresthesias in TOS patients.

Elevated arm stress test (EAST), which was originally described by Roos and Owens in 1966 as a means for eliciting upper extremity

claudication and neurological symptoms. In the test, patients are asked to completely elevate the shoulders and arms (hold-up position) and then to repeatedly clench and unclench their hands . This positioning is designed to constrict the costoclavicular space which will bring weakness and paresthesias in the ulnar and median nerve distributions in patients with TOS within 3 minutes. Attention should also be made to the color of the hands during the EAST, as one may become pale and ischemic if arterial compromise in involved.

The abduction and external rotation test. The arm is abducted, rotated, and held in that position. This test works by a similar mechanism and likewise produces the weakness and numbness seen with EAST in a similar distribution, namely, the C8 to T1 fibers supplying the median and ulnar nerves. In addition, the examiner can sometimes detect a bruit below the lateral portion of the clavicle that is attributable to partial compression of the axillary artery

The “military brace” position can often reproduce TOS symptoms in patients with the disorder. The shoulders are braced backward and downward, effectively causing a narrowing of the thoracic outlet. Symptoms arise within 2 minutes in a susceptible patient. The upper limb tension test uses graduated tension to test for irritation of the brachial

plexus by extending the forearm with the shoulder abducted and externally rotated. Other tests include the timed Morley and Eden studies.

Pulse examination is important to know the arterial involvement. The original Adson's test consisted of assessment of the radial pulse following rotation of the neck to the contralateral side and deep inspiration. Adson's sign is the subsequent loss of the radial pulse. Measurements that quantify blood pressure are more sensitive, and a drop of 20 mm Hg after movement from neutral position or the same pressure difference from one extremity to the other is often present in TOS. These tests are facilitated by using the Doppler probe. None of these aforementioned tests are pathognomonic, but the presence of one or more of them can help support the diagnosis of TOS.

The cervical spine film and chest radiograph are the most important objective studies needed in making the diagnosis of TOS. The bony abnormalities of TOS (cervical ribs, elongated C7 transverse processes, fractures with exostosis or callous formation) can be appreciated on plane chest films. Magnetic resonance imaging is often used in the workup for TOS. Current protocols include measurement of the interscalene triangle and the costoclavicular space, as well as the thickness of the various muscles and any obvious neural of vascular compression.

Computed tomography has also been used to assess for TOS. The exact role for this study has not been determined, but it does appear that provocative maneuvers are beneficial in revealing the anatomical abnormalities.

Somatosensory evoked potentials (SSEPs) can play a role in the workup of TOS. Currently, assessment of the ulnar and median nerves can be used for evidence of their compression at the thoracic outlet. These studies, when abnormal, tend to show lower plexus injury (ulnar) with normal median function. Furthermore, when these patients were studied following operative decompression of their thoracic outlets, more than 90% had correlation between improved symptoms and normalization of their SSEPs .

Arterial Disease

Physical examination can reveal a pulsatile mass in the supraclavicular fossa, but this is not a consistent finding. Clearly, the diagnostic algorithm is not straightforward. Patients may initially be worked up with digital plethysmography or upper extremity duplex examination. These may be abnormal depending on the lesion, and arteriography is almost always required in this setting. When arterial

compression is suspected, attention should first be toward an arch study to include the subclavian and axillary arteries more distally. Often, arterial compression can be better visualized if the arm is abducted 90 degrees, and most studies are obtained with the arms in neutral and abducted positions . When distal embolization is suggested, the angiography should encompass the target sites, often requiring studies of the hand on the affected side.

Surgical management is through, transaxillary first-rib resection followed by arterial repair remains the treatment of choice. If there is a cervical rib, it needs to be removed as well. The area of arterial involvement is usually the retroscalene subclavian to prepectoral region of the axillary artery. Patients presenting with emboli, needs to be treated with catheter-based thrombolysis before ttreating the source.

Aneurysms[3] secondary to TOS usually are treated by, resection and graft reconstruction. Graft can be synthetic material, vein, or arterial grafts. Postoperative anticoagulation is not usually indicated, and patients tend to do well if the thoracic outlet is adequately decompressed and the vessel is no longer subjected to trauma.

An additional consideration in these patients is the presence of reflex sympathetic dystrophy, causalgia, or other autonomic dysfunction. Cervicodorsal or cervicothoracic sympathectomy is offered to these

patients and can often be performed at the time of the arterial repair by standard approaches or by VAT (video assisted thoracoscopic) method.

Finally, patients with arterial consequences of TOS can have diverse complications. Machleder reported an embolizing aneurysm of the posterior humeral circumflex artery necessitating aneurysm excision and cephalic vein bypass of the distal common and posterior humeral circumflex artery to the anterior humeral circumflex artery. Hence the varied problems arising from arterial involvement in TOS leads to the different presentations and concomitantly to the most varied therapies.

Material and Methods

Study Design: Prospective study

Duration : January 2011 to December 2012

Setting: Govt. Stanley Medical College and Hospital, Chennai.

Inclusion Criteria:

1) Patients in the age group between 10 to 80 years
2) Chronic arterial occlusion of upper limb.

Exclusion Criteria:

1) Trauma
2) Aortic Arch Lesions
3) Acute limb ischemia
4) Hemodialysis access ischemic complication

Methodology:

All patients who are admitted with critical upper limb ischemia at the vascular unit were identified from prospectively maintained case sheets and hospital records. Patients were included based upon inclusion and exclusion criteria.

After Documentation of Clinical history, physical examination and evaluation with Doppler and CT Angiogram done for all the patients. Based on the angiographic findings patients were treated by either surgery (open or endovascular) or treated conservatively.

RESULTS

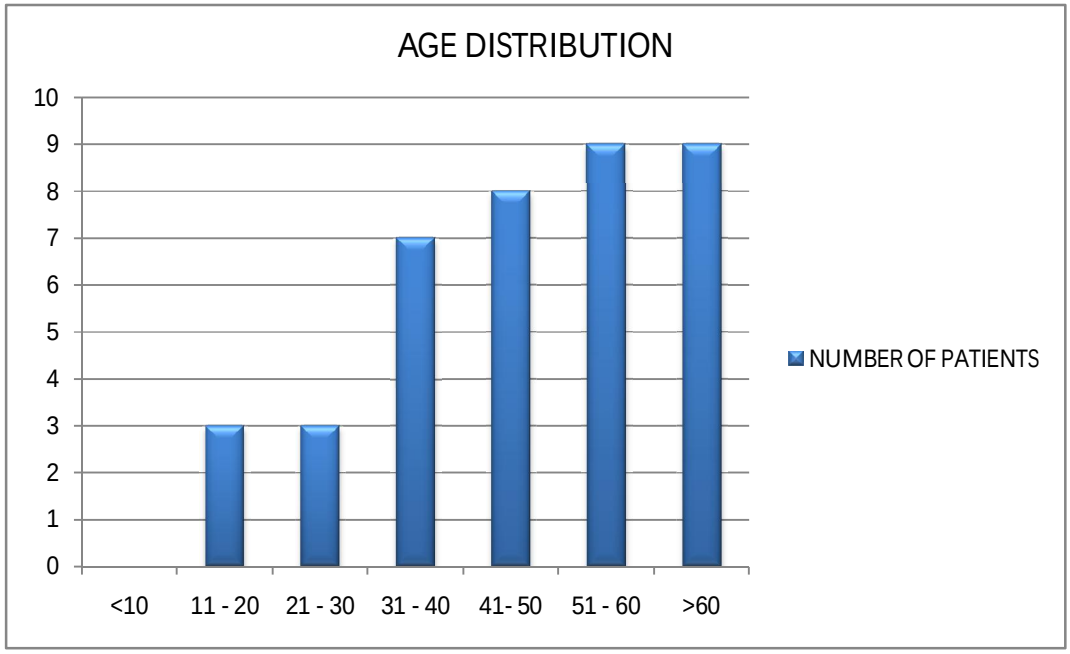
The total number of patients who presented with upper limb ischemia were 39. In this study of the 39 patients 26 cases were male and 13 cases were females. This gives a male to female ratio of 3:2. The predominant cause for upper limb ischemia is due to atherosclerosis which was present in one third of our patients.

Age Distribution

Table 1: Age Group Affected

Age group	Number	%
<10	Nil	Nil
11-20	3	7.69 %
21-30	3	7.69 %
31-40	7	17.95 %
41-50	8	20.51 %
51-60	9	23.07 %
60	9	23.07 %

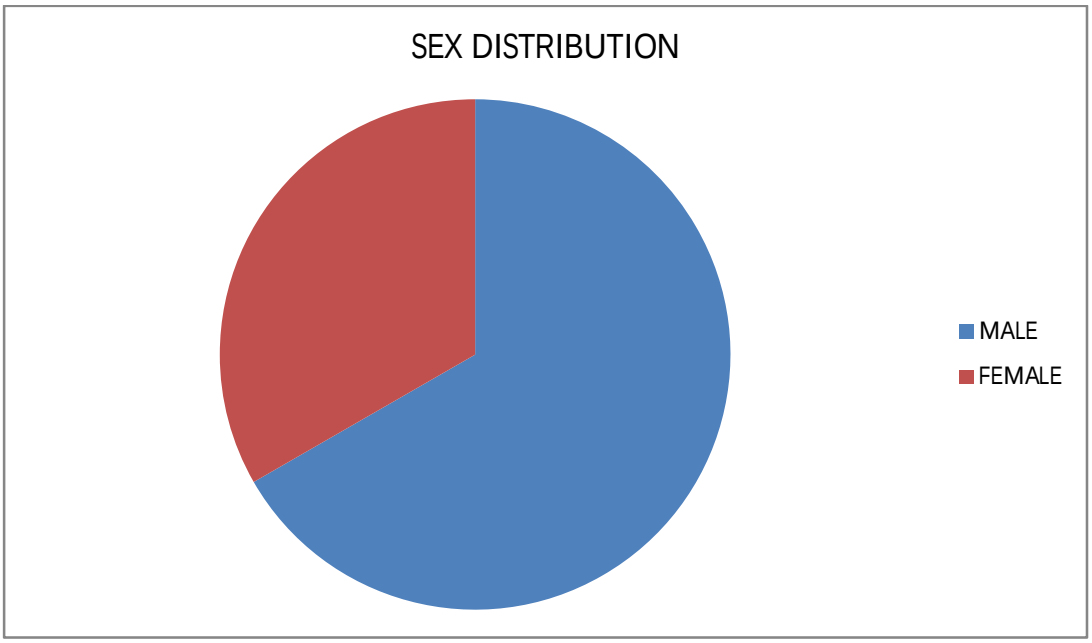
Table 1 shows the age group involved in this study. In this, the majority of patients belonged to more than 50 years and above, followed by patients in the 40-50 years age group.



Sex Incidence

Table 2: Sex Incidence

Gender	No of patients	%
Male	26	66.67 %
Female	13	33.33 %
Total	**39**	



As given in the Table 2 out of 39 cases studied, 26 cases were males, with females accounting for about 13 cases.

Incidence

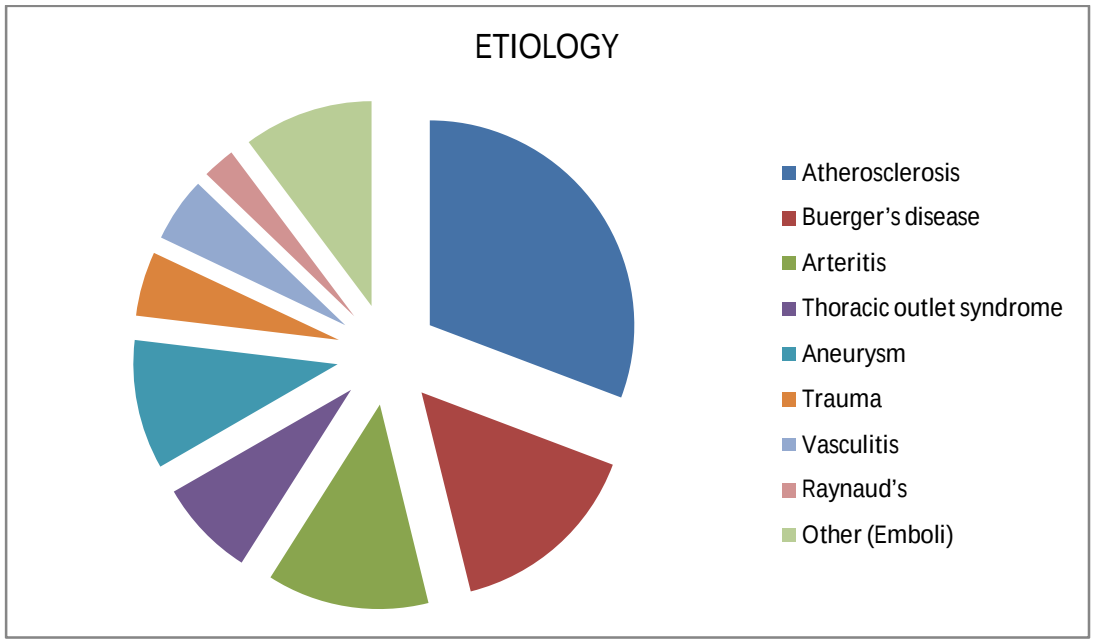
The incidence of Upper Limb Ischemia is more common in Left Upper Limb than Right Side and it accounts for 67 % in our series.

Table : Incidence

Right	Left	Bilateral
8	26	5

Etiology of upper limb ischemia

Etiology	NO. of Cases	%
Atherosclerosis	12	30.76 %
Buerger's disease	6	15.38 %
Arteritis	5	12.82 %
Thoracic outlet syndrome	3	7.69 %
Aneurysm	4	10.25 %
Trauma	2	5.12 %
Vasculitis	2	5.12 %
Raynaud's	1	2.56 %
Other (Emboli)	4	10.25 %



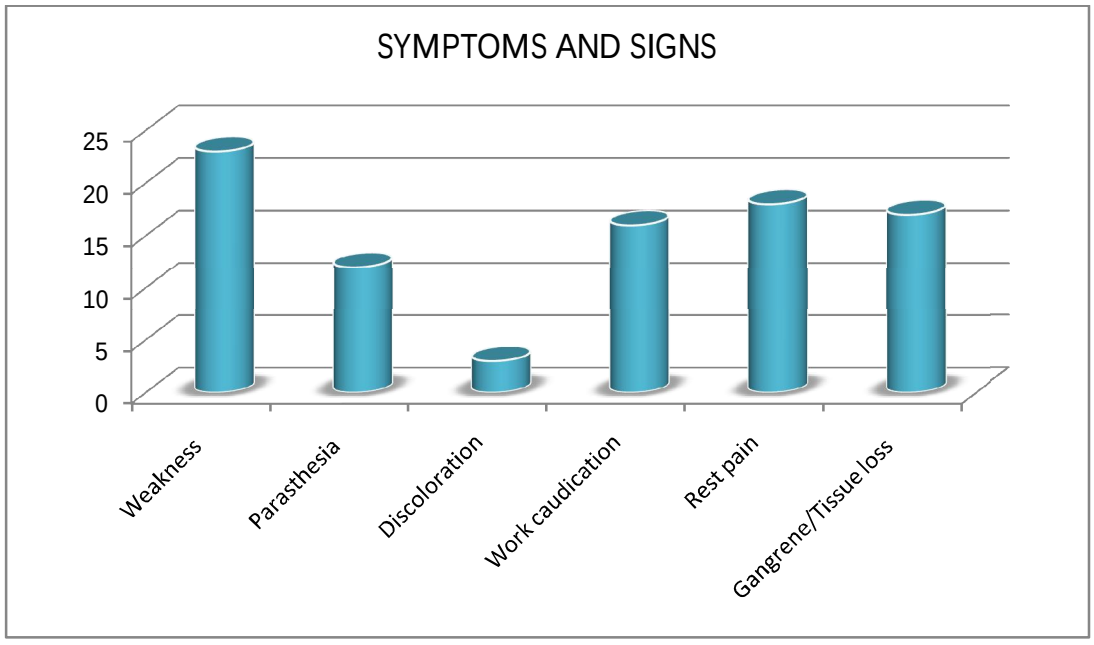
Atherosclerosis constitutes about 30.76% of upper limb ischemia followed by buerger's disease and arteritis which constitutes 15.38% and 12.82% respectively.

Symptoms & Signs

The following table shows the incidence of various symptoms & signs with which the 39 patients studied presented with

Symptoms & Signs	No.of Patients
Weakness	23
Parasthesia	12
Discoloration	3

Work caudication	16
Rest pain	18
Gangrene/Tissue loss	17



Majority of patients presented with weakness and tissue loss.

INVESTIGATIONS

X-ray Cervical spine

X-ray cervical spine both anterior and lateral views was done in all cases to rule out cervical rib. Cervical rib was found in 3 patients.

Feature	No. of Patients
Cervical rib	3

Hand held doppler

Result	No of patients
Pressure difference >10 mmhg	16
No flow	3

All the patients were subjected to hand held Doppler to look for flow in brachial , radial and ulnar arteries. Segmental pressures were measured in all patients and it was significant (pressure difference of more than 10mmhg) were found in 16 patients. Out of 39 patients 3 patients had no flow recorded in radial and ulnar arteries.

Duplex Scan

	No. of cases
Normal	4
Diseased	9
Total	13

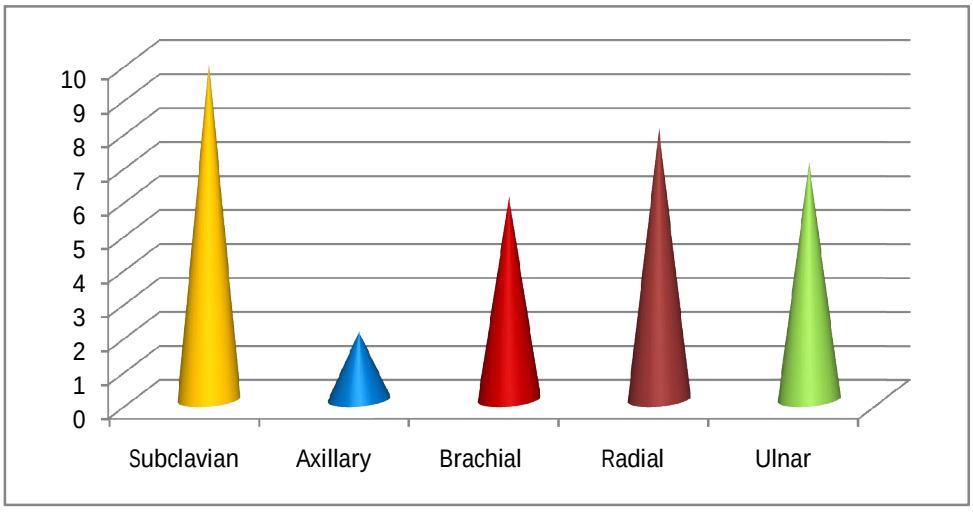
Duplex scan was done in 13 patients. 2 patients had undergone surgery based on Doppler report. 1 patient with an radial artery aneurysm needed further evaluation with angiogram to plan for further management. Except for 4 patients all the patients had disease in brachial, radial or ulnar arteries.

CT Angiogram

A total of 22 patients were subjected to CT angiogram. Out of which 3 patients are evaluated for upper limb ischemia due to aneurysm. 2 patients of traumatic origin and 2 patients of ischemia due to emboli were subjected to angiogram. Out of these 22 patients 13 patients had undergone revascularization procedures after angiogram.

Vessels involved

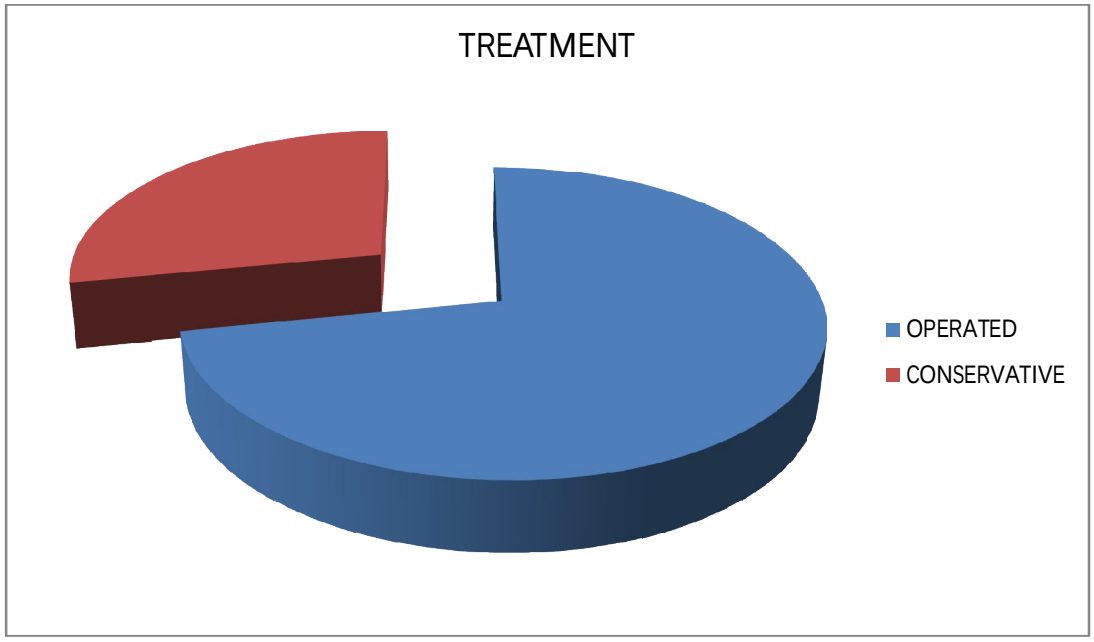
Artery	No. of Patients	Percentage
Subclavian	10	25.64%
Axillary	2	5.12%
Brachial	6	15.38%
Radial	8	20.51%
Ulnar	7	17.95%



In the present series, Subclavian artery is the most commonly involved vessel which accounts for 25.64 %. Radial artery is involved in about 20.51 % of cases. Ulnar artery in about 17.95 % and axillary in 15.38 % of patients.

Ratio Of Revascularisation procedures versus Conservative Treatment

	No of Patients	%
Operated	16	41.02 %
Conservative	23	58.97 %



After a detailed clinical evaluation and suitable investigations, 16 patients underwent revascularization procedures in the form of endovascular procedures or bypass. About 23 patients were managed conservatively with antiplatelets and statins.

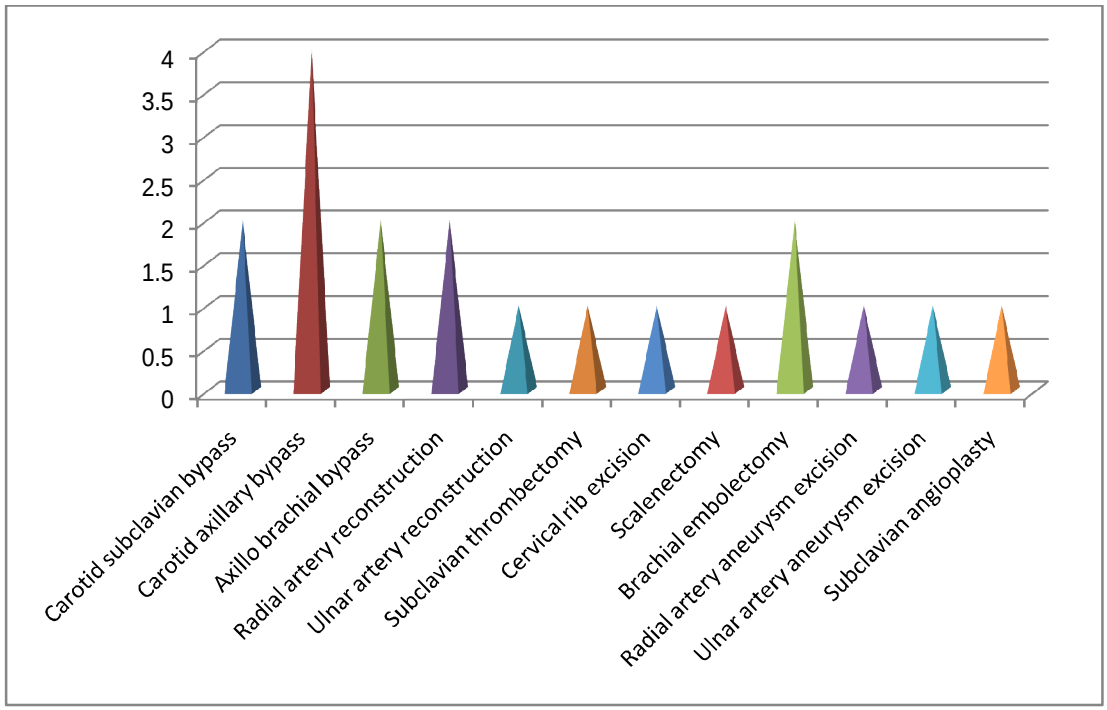
Operative Procedures

The following table shows the various operative procedures carried out on 16 patients with upper limb ischemia. 2 patients of subclavain occlusion had undergone carotid subclavian bypass with PTFE graft. Out of 4 patients who had undergone carotid axillary bypass 3 patients had occlusion of subclaivan artery and 1 patient had occlusion in proximal axillary artery. Of the 2 patients who had undergone axillobrachial bypass one was with reverse long saphenous vein graft other bypass was done using PTFE graft. Radial artery reconstruction using saphenous vein graft was done in 2 patient. 1 patient who had aneurysm of both radial and ulnar artery had undergone reconstruction of radial and ulnar artery using saphenous vein graft.

1 patient of radial artery aneurysm and another patient with ulnar artery aneurysm had undergone excision of the aneurysm. Transbrachial embolectomy was done in 2 patients. Cervical rib excision along with subclavian thrombectomy was carried out in 1 patient. 1 patient was treated with scalenectomy alone.

Endovascular procedure in the form of subclavian angioplasty was done in 1 patient.

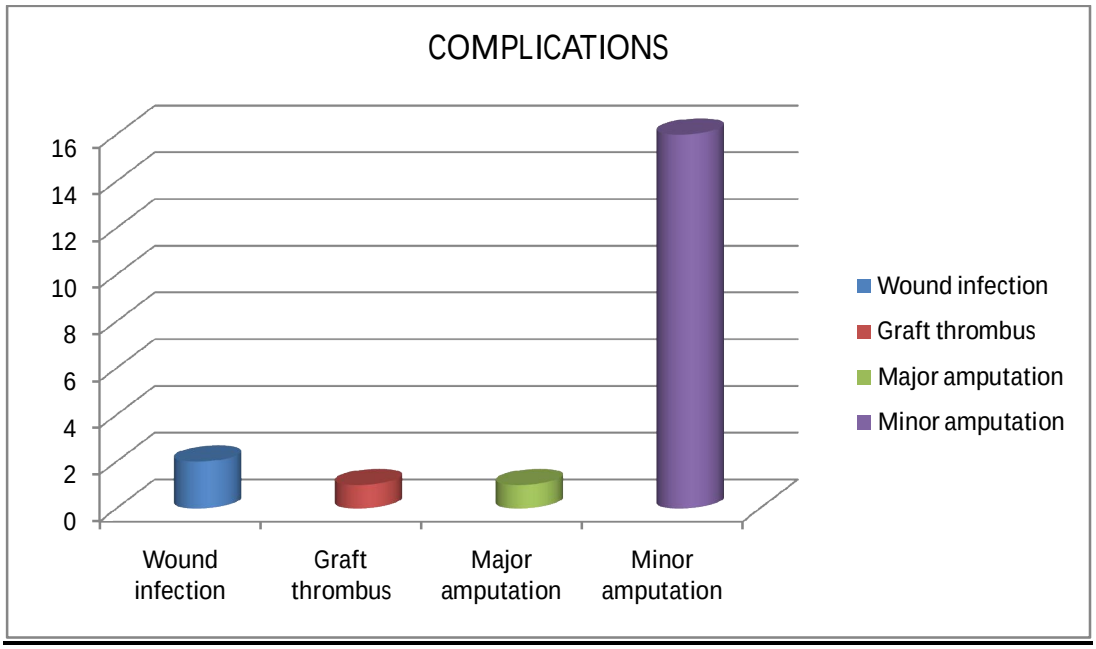
Procedure	Number of Patients
Carotid subclavian bypass	2
Carotid axillary bypass	4
Axillo brachial bypass	2
Radial artery reconstruction	2
Ulnar artery reconstruction	1
Subclavian thrombectomy	1
Cervical rib excision	1
Scalenectomy	1
Brachial embolectomy	2
Radial artery aneurysm excision	1
Ulnar artery aneurysm excision	1
Subclavian angioplasty	1



Post Operative Complication

The following table shows the post operative complications in patients who underwent arterial reconstruction

Wound infection	2
Graft thrombus	1
Major amputation	1
Minor amputation	16



11 patients who had reconstruction in the form of bypass or angioplasty one patient had graft thrombus. 9 patients had regained pulse distally and one patient had flow improvement.

4 patients of upper limb ischemia are due to emboli who presented after 2 weeks. Out of these 2 patients who had undergone embolectomy, 1 patient ended up with above elbow amputation. Minor amputation in the form of phalanx amputation which were gangrenous were carried out in 16 patients.

Morbidity & Mortality

The Mean range of stay of patients in the hospital ranged from 11-15 days.

The range varied from 0 days to 29 days.

The following table shows duration of hospital stay of patients with upper limb ischemia.

Duration (days)	No. of Patients	Percentage
0-5	5	12.82
6-10	10	25.64
11-15	18	46.15
16-20	3	7.69
21-25	2	5.12
25 -29	1	2.56

There is no mortality in the present series.

Discussion

This two year study included 39 patients out of which 16 patients had undergone surgical treatment for salvaging upper limb. About 46% of patients were in the age group above 50 years. The majority of patients presented due to atherosclerotic[1] occlusion (30.76%). Four patients presented due to complication of aneurysm of radial and ulnar arteries. A further four patients with chronic upper limb ischemia had a delayed presentation following Thromboembolism. Subclavian artery occlusion (25.64%) is the most common vessel involved.

Atherosclerotic occlusive disease

Twelve patients are diagnosed with atherosclerosis. The mean age was 59 years, Of which eight were male and four female. One patient had undergone carotid subclavian and another carotid axillary bypass for subclavian occlusion disease. One patient was treated with subclavian angioplasty for subclavian stenosis.

All the patients who had undergone revascularization by means of bypass or angioplasty had regained distal pulses. One patient who presented with occlusion of brachial artery with no reformation distally ended up with above elbow amputation

Patients with successful revascularization for chronic ischemia were followed up regularly. All grafts were patent clinically or on duplex ultrasonography .The patency rate for upperlimb bypass was 100%.

Buerger's Disease

Six patients presented with upper limb ischemia due to thromboangitis obliterans. All the patients were smokers and all were male.

One patient had disease progression upto brachial artery hence was treated with Axillo brachial bypass[16]. Post operatively he had palpable radial pulse. Other five patients were evaluated with duplex scan and were treated with antiplatelets. Three patients who had distal phalanx gangrene had undergone amputation of the gangrenous part. All the patients were advised to stop smoking.

Arteritis

Five patients were diagnosed to have arteritis in our series. Out these five patients four were female. All the patients were in the 20-40 years age group. With one patient who was 45 years of age. All the patients had muscle wasting with weakness of upper limb. Three patents also had work claudication.

All the five patients had raised erythrocyte sedimentation rate and C-Reactive protein levels at the time of diagnosis. Hence they were treated with steroids . Four patient who had clinically subclavian occlusive disease were evaluated with CT Angiogram. After decreasing the inflammatory markers[15] to optimal level. One patient was treated with carotid subclavian and the other with carotid axillary bypass with PTFE graft. One patient had undergone axillobrachial bypass. One patient who had subclavian disease did not agree for any intervention hence was treated with medications.

Thoracic Outlet Syndrome

Three patients two female and one male presented with upperlimb ischemia due to arterial involvement. One patient underwent revascularization procedures for ischemia of the upper limb in the form of cervical rib resection with subclavian thrombectomy. Other patient was treated with scalenectomy[14] alone. The third patient who had claudication with pressure difference of 10mmhg was only 17 years of age hence was differed surgery . She was advised to do exercise of shoulder movements and to follow up regularly.

Aneurysm

Aneuysm is the cause of upper limb ischemia in four patients. All the four were male. Two patients were evaluated with CT angiogram, one patient who had aneurysm of radial artery at the snuff box was diagnosed clinically. Other patient had ulnar artery aneurysm at the hypothenar level was evaluated with duplex scan.

One patient had both radial and ulnar atery aneurysm hence had undergone revascularization of both arteries with saphenous vein graft. The other radial artery aneurysm patient had undergone excision with reconstruction. One patient who had aneurysm at the snuff box was treated with excision of the aneurysm alone. The fourth patient with ulnar artery aneurysm was treated similarly with excision alone.

Traumatic Injuries

Two male patient presented with weakness, muscle wasting and claudication six months after trauma to the upper limb from road traffic accident. Both these patients had absent upper limb pulses. Both patient also had associated brachial plexus injury. hence were evaluated with CT Angiogram which showed occlusion of subclavian artery.

Both patient had undergone carotid axillary bypass with PTFE graft. Both patient who had concomitant brachial plexus injury had undergone neurolysis for the same. One patient also had subtotal resection of clavicle for exposing the brachial plexus. Both patient had regained distal pulse .

Vasculitis and Raynaud's Disease

Two patient presented with upperlimb ischemia due to vasculitis. Both these patients were femle and were diagnosed with SLE and were positive for antinuclear antibody and double stranded DNA. They presented with digital ulcer. They also had palpable radial and ulnar pulse. Hence were treated with antiplatelets alone.

One patient presented due to primary Raynaud's with discoloration on and off. Hence was advised with reassurance and to avoid cold exposure.

Thromboembolism

Thromboembolism was the etiologic factor in four patient. Three of them were male and one female patient. All the patients presented two weeks after occurrence of pain.

All four patients were evaluated with echocardiography, but only three patients had detectable cardiac abnormalities in the form of

hypokinesia of inferior wall . These patients also had ejection fraction of less than 40%. But no mural thrombus was found in any of these patients

Two patient had undergone transbrachial embolectomy[15] with fasciotomy and had flow improvement post operatively. Limb was salvaged in all the patients with minor amputations involving the digits.

Conclusion

1. Though incidence is less, the upper limb critical ischemia can result in severe functional impairment and disability.

2. Presentation of critical limb ischemia of upper limb results in functional loss than compared to lower limb ischemia because of damage to collaterals hence early revascularisation is essential.

3. CT or MR Angiogram provides adequate information to plan for revascularisation in upper limb ischemia and this obviates the need for Digital subtraction angiography which is invasive with more procedural complications.

4. In the presence of proximal artery occlusion like subclavian, axillary Ipsilateral carotid serves as a good donor vessel, thus obviating the need for cross over from opposite side.

5. Though it is a uncommon entity detailed history taking, physical examination with prompt recognition of upper limb ischaemia and active approach to management in the form of Angiogram and Revascularisation procedures are crucial in obtaining a good outcome and reducing the risk of late disabling effects .

6. The principle of revascularisation is to preserve the limb if not possible to preserve maximum length for effective fixing of prosthesis.

7. Distal bypass to radial or ulnar artery are equally effective as compared to lower limb

8. Patency rate of 94% for upper limb bypass irrespective of the etiology.

9. Early intervention results in good functional limb

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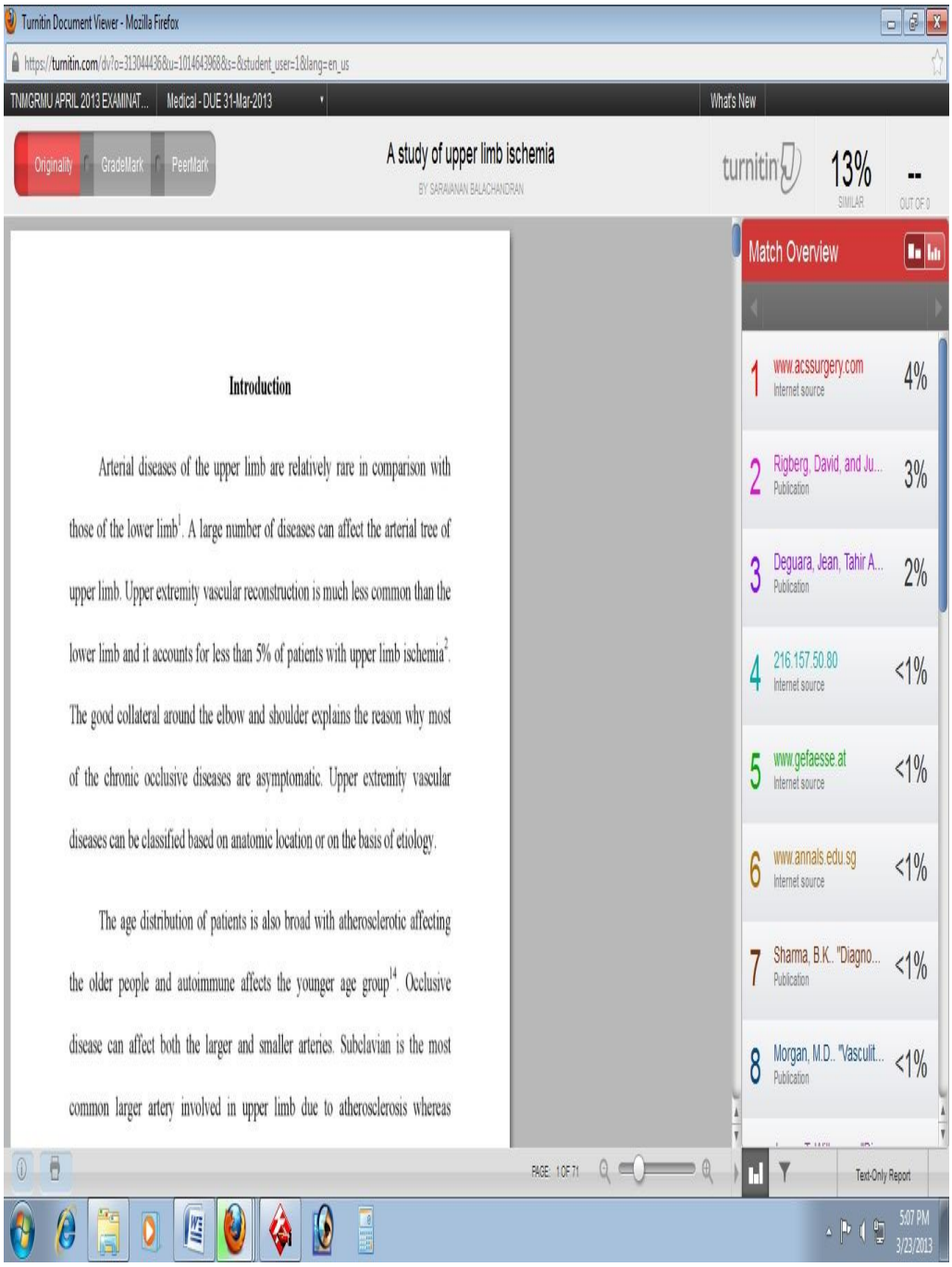
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A study of upper limb ischemia
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Introduction
Arterial diseases of the upper limb are relatively rare in comparison with those of the lower limb[1]. A large number of diseases can affect the arterial tree of upper limb. Upper extremity vascular reconstruction is much less common than the lower limb and it accounts for less than 5% of patients with upper limb ischemia[2]. The good collateral around the elbow and shoulder explains the reason why most of the chronic occlusive diseases are asymptomatic. Upper extremity vascular diseases can be classified based on anatomic location or on the basis of etiology.
The age distribution of patients is also broad with atherosclerotic affecting the older people and autoimmune affects the younger age group[14]. Occlusive disease can affect both the larger and smaller arteries. Subclavian is the most common larger artery involved in upper limb due to atherosclerosis whereas
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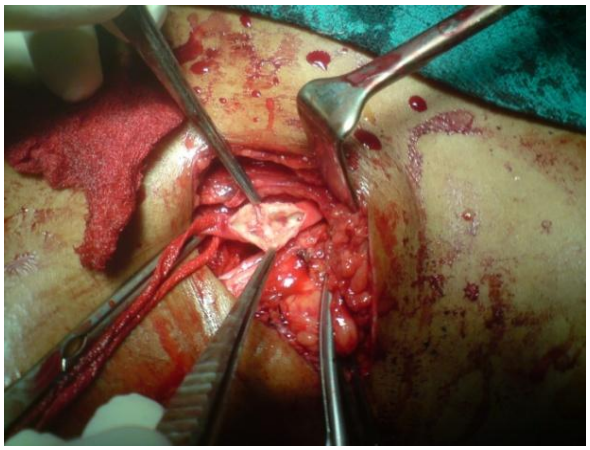
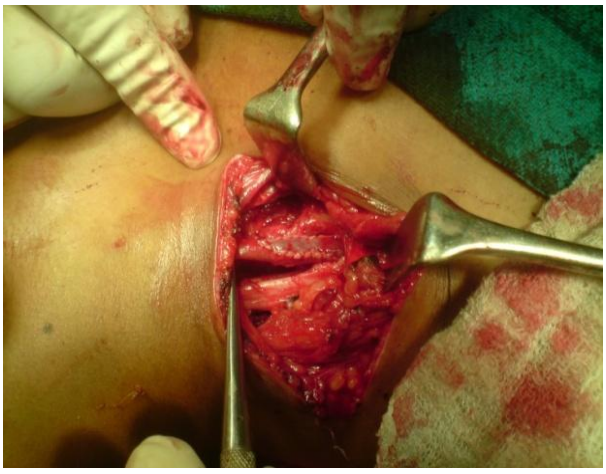
Text-Only Report
5:07 PM
3/23/2013

S.NO	Name	Age	Sex	I.P No	Diagnosis	Treatment
1.	Palani	61	M	7033	ulnar art aneurysm –Rt side	excision
2.	Sankaralingam	68	M	1246	Radial and ulnar artery aneurysm- Lt side	Radial and ulnar reconstruction
3.	Jaya	35	F	13592	Subclavian occlusion-Left Side	Carotid axillary bypass
4.	Pandian	45	M	9355	Brachial occlusion-rt side	Thrombectomy
5.	Manoharan	45	M	6365	Lt subclavian occlusive disease	Carotid subclavian bypass
6.	Tamilarasi	45	F	35362	Lt subclavian occlusive disease	Conservative management
7	Delliammal	65	F	45676	Rt Brachial disease	Conservative management
8.	Marutham	60	M	7673	Lt Brachial disease	Conservative management
9.	Banumathi	58	F	11325	SLE with Vasculitis-Bilateral	Medications
10.	Jasmine	17	F	19255	B/L cervical rib	Reassurance and follow up
11.	Jerina	40	F	32117	Arteritis-Right Side	Medications
12.	Nagarathinam	65	F	3289	Lt Axillary occlusion	Above elbow amputation
13.	Vani	35	F	41876	Vasculitis with gangrene index finger-lt side	Finger amputation
14.	chinammal	70	F	44922	Vasculitis Lt UL	medications
15.	Devi	30	F	141/12	Lt cervical rib with index finger gangrene	Scalenectomy
16.	Babyammal	70	F	5986	Brachial UEAD-Right Side	Embelectomy
17.	Malliga	60	F	7123	Lt UEAD with Gangrene …. Index and little finger	Thrombectomy
18.	Raman	55	M	37047	Lt Radial Artery pseudo Aneurysm	Excision done
19.	Theliyappan	60	M	40261	Left subclavian occlusive disease	Conservative management
20.	Dhivakaran	70	M	52611	Lt UEAD with Lt index finger gangrene	Embolectomy
21.	Raji	50	M	54856	Rt Radial/ulnar disease	Conservative management
22.	Chandra sekar	43	M	3943	TAO with Rt index finger gangrene	medications

S.NO	Name	Age	Sex	I.P No	Diagnosis	Treatment
23.	Murugesan	41	M	16864	Subclavian Occlusive Disease	Carotid axillary bypass with Brachial neurolysis
24.	Elangovan	48	M	25086	Upper limb TAO	Medical management
25.	Muthu	24	M	42115	Rt Subclavian occlusive disease with brachial plexus injury with fracture humerus	Conservative Management
26.	Siva	22	M	29109	Arteritis – Left Side	Axillo – Brachial bypass with PTFE graft
27.	Muniyappan	50	M	33600	Lt upper limb TAO	medications
28.	Karuna	40	M	38002	Lt Brachial Occlusive Diseasae	Trans brachial embelectomy
29.	Nagaraj	46	M	5985	Lt subclavian occlusive disease	Lt carotid axillary bypass
30.	Joel madan	20	M	7390	Lt Radial artery aneurysm	Excision and radial artery reconstruction
31.	Karthikeyan	55	M	7750	Lt subclavian occlusive disease	medications
32.	Radhakrishnan	39	M	10064	TAO – Left Side	Lt Axillo Brachial Bypass
33.	Nataraj	60	M	11521	Atherosclerotic occlusive disease- Left side	medications
34.	Balaraman	31	M	20421	Lt brachial artery occlusive disease	Conservative Management
35.	Vijayakumar	19	M	44443	Subclavian occlusive disease	Lt carotid axillary PTFE bypass with left subtotal excision of clavicle with Neurolysis of Brachial plexus
36.	R.S. Singh	82	M	46143	RAYNALD'S disease Left upper limb	medications
37.	Perumal	42	M	2275	TAO Left upper limb	medications
38.	Parasuraman	55	M	3189	Cervical Rib - Left side	Cervical rib excision with Subclavian Thrombectomy
39.	Alagumani	26	F	23011	Subclavian occlusion	Carotid Subclavian bypass



Subclavian Thrombectomy



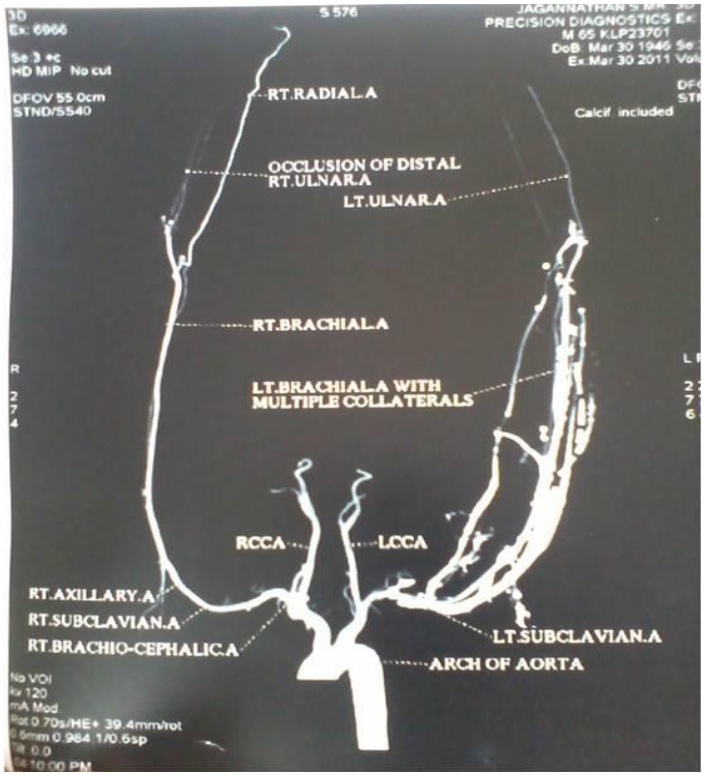
Subclavian Tailoring & Patch Plasty

Cervical Rib

Angiogram showing Right Subclavian Occlusion

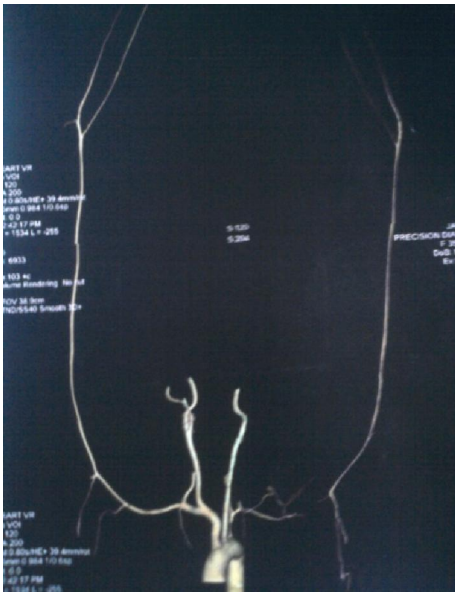
Carotid Subclavian Bypass – Post op Picture

Angiogram showing Right Subclavian occlusion-Post Traumatic



Angiogram showing occlusion of Left Radial & Ulnar artery

Subclavian occlusion – Takayasu's Arteritis



Carotid Axillary Bypass with PTFE Graft

Carotid Axillary Bypass – Post op Picture

PROFORMA

Case no :

Name: _____ IP. No: _____

Age: _____ Sex: _____Occupation:_____

Address:_____

Contact No: _____

Date of Admission: Date of Discharge: Date of Surgery :

Presenting History :

Work Claudication Duration Onset

Discoloration Duration Onset

Past History:

- Drug intake
- Tobacco use (Smoking, Snuff, chewing)
- Ischemic heart disease
- known DM/HT/PTB

General Examination:

Vascular Examination:

Investigations :

Hb %,

ESR

Lipidogram

Immunology profile:

- Rheumatoid factor
- C-Reactive protein
- Aso Titre

Segmental Pressures

X Ray Cervical Spine

Duplex Scan

CT Angiogram

Management

Surgical Procedure

Post operative status

Morbidity

Mortatility